ABC of intensive care
Neurological support

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The neurological conditions that require management in intensive care are diverse. Indications for admission range from maintaining the airway to control of seizures and intracranial pressure. Intensive care of a patient with a neurological disease requires a partnership between the referring specialist and intensive care doctors. Despite the diversity of the neurological diseases being managed some standard principles apply.

Standard principles for neurological intensive care

- The airway should be protected, generally with an endotracheal tube or tracheostomy
- Normal gas exchange should be maintained using mechanical ventilation if necessary. Especially in conditions of critical cerebral oxygen supply—for example, acute brain injury— the arterial oxygen tension (Pao_2) should be kept above 12 kPa and the arterial carbon dioxide tension ($Paco_2$) at low normal values (4.0-4.5 kPa)
- Maintenance of an adequate cerebral perfusion pressure is essential to maintain cerebral oxygen delivery
- Specialised measurement techniques such as monitoring intracranial pressure assist management

Acute brain injury and encephalopathy

Patients with acute brain injury, regardless of the cause, all raise similar intensive care problems. Some care, including ventilation, control of intracranial and cerebral perfusion pressure, and anticonvulsant treatment, may be similar, although patients will also require specific treatment of their condition. Patients should have their pupil size and responses assessed and conscious level measured by the Glasgow coma scale. These signs should be reassessed regularly thereafter.

Causes of acute brain injury

- Trauma
- Aneurysmal subarachnoid haemorrhage
- Ischaemic or haemorrhagic stroke
- Infection (encephalitis or meningitis)
- Vasculitis (such as systemic lupus erythematosus)
- Demyelination (such as acute demyelinating encephalomyelitis)
- Tumour or peritumoral haemorrhage

Aims of intensive care management

The number and duration of secondary insults affect outcome. In particular, hypotension, decreased cerebral perfusion pressure, hypoxaemia, and hyperthermia are associated with a worse outcome. Intensive care management aims to avoid secondary insults and to optimise cerebral oxygenation by ensuring a normal arterial oxygen content and by maintaining cerebral perfusion pressure above 70 mm Hg. This figure may be modified depending on the jugular bulb oxygen saturation. Intracranial pressure should generally be below 25 mm Hg.

Intubated patients need sedation to avoid rises in intracranial pressure. Brain injured patients are prone to early nosocomial chest infection, due to impaired upper airway reflexes, and broad spectrum antibiotic prophylaxis may be advisable.

General aspects of neurointensive care

- No parenteral non-ionic fluid must be given
- Keep plasma sodium concentration >140 mmol/l. A fall produces an osmotic gradient across the blood-brain barrier and aggravates cerebral oedema
- Avoid hyperglycaemia and hypoglycaemia. Hyperglycaemia may aggravate ischaemic brain injury by increasing cerebral lactic acidosis. Blood glucose levels >11 mmol/l should be treated
- Feed through an orogastric tube. Gastric motility drugs can be given as required
- Anti-thromboembolism stockings; avoid low dose heparin
- 15-30° head up tilt with the head kept in a neutral position may improve cerebral perfusion pressure

Sedation and paralysis

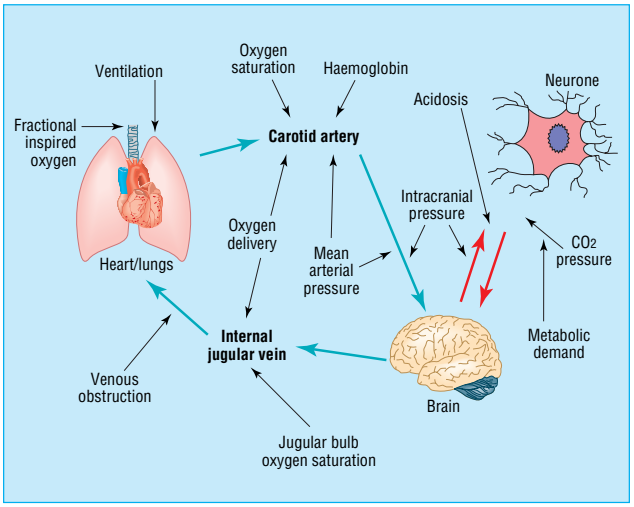
Sedation is required to depress coughing and spontaneous respiratory efforts in response to intubation and ventilation. Sedation depresses the cerebral metabolic rate and may improve the cerebral oxygen supply:demand ratio. A benzodiazepine (midazolam) is usually infused in combination with a short acting opioid such as alfentanil. Intravenous propofol can be used for depression of the cerebral metabolic rate, which coupled with cerebral vasoconstriction reduces intracranial pressure. However, it may also substantially reduce mean arterial pressure.

Patients with severe head injury generally require neuromuscular paralysis for the initial 12-24 hours in intensive care to prevent uncontrolled rises in intrathoracic and hence intracranial pressure. Thereafter, relaxants can be allowed to wear off. If patients remain well sedated without a rise in intracranial pressure they may be left unparalysed.

Specific monitoring techniques

The final common pathway in all acute brain injury is thought to be failure of oxygen delivery—that is, ischaemia. Monitors have been developed to detect critical falls in oxygen delivery.

Intracranial pressure monitoring—Most centres now use intraparenchymal monitors that are usually placed into the right (non-dominant) frontal region through a small burr hole.



Interdependence of systemic and cerebral oxygen delivery variables

Although the intracranial pressure is important (normally <10 mm Hg, acceptable upper limit 25 mm Hg), the cerebral perfusion pressure is more important. It is calculated as mean arterial pressure minus intracranial pressure. Cerebral perfusion pressure is the principal determinant of cerebral blood flow.

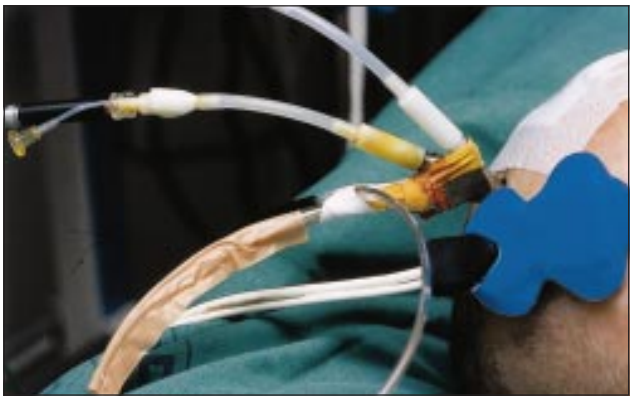
Jugular bulb oxygen saturation monitoring—Bedside measurement of cerebral blood flow is difficult, but the jugular bulb oxygen saturation (Sjo_2) gives an indication of cerebral blood flow in relation to cerebral metabolic oxygen demand. The normal range is 50-75%. Low values indicate increased oxygen extraction, possibly due to low cerebral perfusion pressure or hyperventilation, while high values indicate cerebral hyperaemia. Monitoring jugular bulb oxygen saturation allows assessment of the effect of interventions on cerebral perfusion.

Transcranial Doppler ultrasound through a "window" in the temporal bone can be used to measure blood flow velocity in the basal cerebral arteries. The technique gives an indication of cerebral perfusion pressure and the presence of cerebral vessel narrowing if extracranial internal carotid velocities can be assessed (Lindegaard index).

Brain tissue oxygenation ($PBro_2$)—Regional estimates of oxygen pressure obtained by miniature Clark electrodes placed within cerebral tissue have been shown to correlate with outcome.

Processed electroencephalographic monitoring—Full electroencephalographic monitoring is generally too complex for routine use in intensive care. Various methods of electroencephalographic processing exist to allow assessment of cerebral electrical activity, detection of seizures, and titration of barbiturates or other anaesthetic treatment. Excessive anaesthetic infusion results in an isoelectric "flat" trace.

Solid state intraparenchymal intracranial pressure monitor



Brain tissue oxygenation, temperature, and pressure are measured by three probes through one burr hole. A near infrared spectroscopy optode is placed on the frontal region of the scalp and insulated from incident light

All neurological intensive care units require 24 hour access to computed tomography

Cerebral protection

Considerable effort and funding have gone towards developing a neuroprotective drug to reduce mortality after brain trauma and improve functional recovery. There have been many failed or inconclusive studies, and the future of pharmacological neuroprotection after traumatic brain injury is in doubt. There is no evidence to support the use of corticosteroids after traumatic brain injury or routine use of profound hyperventilation ($Paco_2$ <3.3 kPa). At best, these treatments do no good, and they may adversely affect outcome.

Clinicians managing patients with a head injury are therefore left with detection and prevention of secondary insults to the brain, including management of medical complications of brain injury and non-pharmaceutical interventions that might improve the brain's response to trauma. Of the potential interventions, moderate hypothermia is the most promising.

Indications for intensive care for patients with head injury

- Not obeying commands after resuscitation and before intubation and ventilation or neurosurgical intervention
- Associated chest injury or multiple injuries that prevent continued assessment of head injury
- Unable to maintain airway or gas exchange
- Spontaneous hyperventilation ($Paco_2$ <3.5 kPa)
- Repeated seizures

Subarachnoid haemorrhage

The outcome from severe subarachnoid haemorrhage has been poor. An aggressive approach based on intensive care and early surgical or endovascular intervention may improve outcome.

In general, principles of management are similar to those in traumatic brain injury, although specific management may be required for neurogenic pulmonary oedema—for example, pulmonary artery catheterisation and inotrope therapy. Patients with acute hydrocephalus require early drainage. Cerebral angiography and surgical clipping or coil embolisation should be considered early after cardiovascular control and adequate oxygenation have been achieved.

Indications for intensive care in subarachnoid haemorrhage

- Poor grade aneurysmal subarachnoid haemorrhage
- Rapidly decreasing Glasgow coma score or focal neurological deficit
- Complications, including cardiorespiratory dysfunction and particularly neurogenic pulmonary oedema (characterised by acute, severe, but reversible left ventricular dysfunction with associated pulmonary oedema)
- Delayed neurological deficit

Delayed neurological defict

Systemic therapy—All patients with subarachnoid haemorrhage should receive the calcium channel blocker nimodipine; however, caution is needed in haemodynamically

unstable patients. In addition, patients should be actively hydrated; a combination of hypervolaemia, hypertension (using noradrenaline or dopamine), and haemodilution may reverse delayed neurological deficit. Early definitive treatment of the aneurysm—that is, before 96 hours—allows induction of hypertension without the risk of rebleeding.

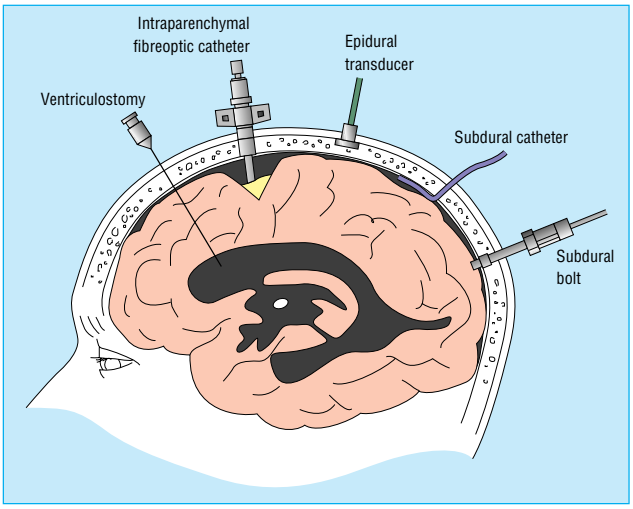
Local therapy—Re-angiography and treatment for local arterial narrowing may be considered. Papaverine, nitroprusside, angioplasty, and thrombolysis have all been successful.

Duration of intensive care

In general, sedation and ventilation are maintained for at least 48 hours after brain injury, by which time evidence of brain swelling will be present. Ventilation should be continued until interventions to control intracranial pressure have not been needed for 24 hours. This may take 10-14 days of intensive care.

Acute ischaemic stroke

Increasing public awareness and early computed tomography will allow more aggressive management including thrombolysis and, in selected cases, intensive care.



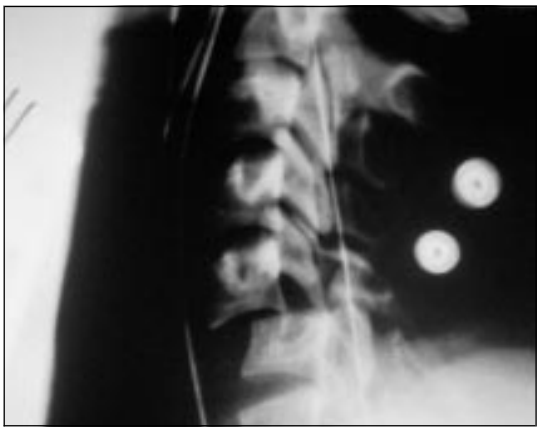
Sites of measurement of intracranial pressure

Spinal cord injury

Physiological regulation of blood flow in the spinal cord is the same as for cerebral blood flow. Thus, most of the principles for managing traumatic brain injury apply to spinal cord injury.

Initial injury is associated with haemodynamic instability and cardiac arrhythmias, reportedly because of sympathetic stimulation. This is followed by the sudden onset of hypotension; loss of vasomotor tone is compounded in lesions above T2-T5, when sympathetic outflow to the heart is lost and parasympathetic tone is unopposed. The result is cardiac dysfunction, hypotension, and bradycardia. Spinal shock can last from weeks to months and is best managed in experienced intensive care units. Once reflex activity has returned below the level of the lesion autonomic dysreflexia can occur. Improvements in initial resuscitation, with early administration of high dose methylprednisolone, have improved functional recovery.

Perfusion pressure (mean arterial pressure minus cerebrospinal fluid pressure) is the main determinant of blood flow in the spinal cord



Lateral radiograph of unstable neck injury

Peripheral neuropathies and neuromuscular and muscle disorders

The main conditions for which patients require intensive care are Guillain Barré syndrome and myasthenia gravis. Patients with other motor neuropathies, polymyositis, and muscular dystrophies may also require admission. Most patients are referred to intensive care with acute respiratory failure.

Patients at risk of developing respiratory failure should have pulse and respiratory rate measured hourly together with regular observation of chest movements and air entry and assessment of vital capacity. Pulse oximetry is useful when supplemental oxygen therapy is unnecessary, but a fall in oxygen saturation is a late sign in patients receiving oxygen. Patients with a vital capacity <1.5 litre need their arterial blood gases checked. A vital capacity <1 litre implies an inadequate cough.

Endotracheal intubation is indicated when impaired airway control (either as a result of bulbar dysfunction or inadequate cough) leads to an increased risk of aspiration. Most patients requiring intubation will need ventilation, as will patients with hypoxaemia or hypercapnia.

Mortality from Guillain Barré syndrome in intensive care is 3-8%, mainly because of avoidable complications. Problems include autonomic neuropathy, sepsis, constipation, deep

Causes of acute respiratory failure in peripheral neurological disease

- Global respiratory muscle failure leading to inadequate alveolar ventilation and hypercapnia
- Fall in vital capacity due to muscle weakness resulting in failure to cough and clear secretions. This may cause acute respiratory failure due to bronchial obstruction and lobar or segmental collapse
- Bulbar dysfunction leading to failure of swallowing and coughing with consequent aspiration

Useful drugs for intubated patients with peripheral neuromuscular problems

Problem	Suggested treatment
Anxiolysis	Nasogastric diazepam
Neuropathic pain	Amitriptyline or carbamazepine
Musculoskeletal pain	Non-steroidal anti-inflammatory drugs
Initial artificial airway discomfort	Morphine

venous thrombosis, and depression. Scrupulous infection surveillance and careful electrocardiographic and haemodynamic monitoring are therefore essential.

Critical illness polyneuropathy
Critical illness polyneuropathy is a potential complication in patients with sepsis and multiple organ failure. It can result in areflexia, gross muscle wasting, and failure to wean from the ventilator. It therefore prolongs the period of intensive care.

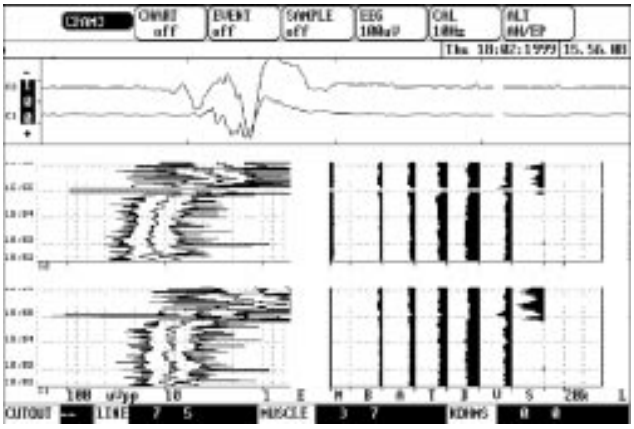
Seizures

Prolonged or recurrent tonic-clonic seizures persisting for over 30 minutes (status epilepticus) constitute a medical emergency and require rapid treatment. Failure to control the seizures will result in massive catecholamine release, hypoxaemia, increased cerebral metabolism, hyperpyrexia, and hyperglycaemia.

Most patients respond to standard treatment—that is, oxygen, airway maintenance, and intravenous diazepam 5 mg, repeated if required. The cause of the seizures should be pursued and treated when appropriate—for example, glucose, calcium, or high dose vitamin B. If the seizures are not controlled with diazepam, or the patient develops hypoxaemia or loss of airway integrity, intravenous anaesthesia, endotracheal intubation, and ventilation are required. Thiopentone is the standard anaesthetic and is titrated until a burst suppression pattern is seen in processed electroencephalograms. Propofol is an alternative. It has the advantage that consciousness rapidly returns after it is stopped because it is quickly metabolised. Patients not already receiving therapeutic doses of phenytoin or other anticonvulsants should be loaded and the propofol or thiopentone dose maintained until therapeutic levels are achieved.

Effects of persistent seizures

- Cerebral and systemic hypoxia
- Lactic acidosis
- Neurogenic pulmonary oedema
- Rhabdomyolysis
- Hyperkalaemia
- Renal failure
- Hepatic necrosis
- Disseminated intravascular coagulation



Processed electroencephalograph of patient with burst suppression pattern

Outcome

Traumatic coma
Doctors and families of patients in coma face a difficult decision when considering whether life extending care will achieve a desirable outcome. Functional recovery can be assessed using the five point Glasgow outcome scale or the more detailed SF-36 health survey questionnaire. Sophisticated measures of functional recovery have also been developed.

Non-traumatic coma
Development of out of hospital resuscitation and improved training of paramedical staff have resulted in an increase in the number of patients in coma after cardiac arrest. Reliable prognosis can be achieved by assessing five variables in the first 3 days after insult. Abnormal brain stem reflexes and absent motor response best predict functional outcome.

Main determinants of outcome of traumatic coma

- Age
- Glasgow coma score after resuscitation
- Computed tomographic diagnosis
- Brain stem responses (pupil reaction)
- Presence of hypotension and hypoxia

Variables for assessing outcome of non-traumatic coma

- Abnormal brain stem responses
- Absent withdrawal response to pain
- Absent verbal response
- Plasma creatinine concentration >132.6 μmol/l
- Age ≥70 years

Patients with 4-5 of these risk factors at 72 hours have a 97% mortality at 2 months

After intensive care

Most patients require extra nursing, medical, and paramedical support after intensive care. A diminished level of consciousness or irritability in patients who have had acute brain injury may make nursing difficult. A tracheostomy is often required for aspiration of tracheobronchial secretions, while continuous positive airways pressure is needed to maintain basal lung expansion in the absence of spontaneous large tidal volume sighs. In patients with impaired consciousness and bulbar dysfunction, a percutaneous endoscopically guided gastrostomy may help feeding. All patients require a huge input from physiotherapists, speech therapists, occupational therapists, and nurses for full rehabilitation.

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